found in the bladder or kidneys, but the patients experienced almost immediate relief from their bladder irritation following such examination. Ordinarily I have not considered these cases as organic strictures, but rather thought them to be abnormally small urethras. Considering Stevens' findings and reports, these cases are undoubtedly organic contractions following either infections or trauma.

I feel both complimented and benefited in having this opportunity of reviewing this very complete essay.

HERBERT AUGUSTUS ROSENKRANZ, M. D. (W. P. Story Building, Los Angeles)—I have been much impressed with the system and completeness of Doctor Stevens' paper, which bears evidence of many revisions. One might compare it in this respect with the shorter stories of Balzac which, through their masterful technique, betray the many revisions and hard work that preceded the boiled-down, finished classic.

This paper is particularly timely, since we as urologists are too prone to concentrate on searching the kidneys and their immediate adnexae for abnormalities to the exclusion of the urethra in the female, although all of us learned early in our careers the folly of doing a male cystoscopy without having at hand also the complete urethroscopic armamentarium. The female urethra has been neglected urethroscopically and urethrometrically. Some practitioners for many years routinely dilated the female urethra for symptoms of irritable bladder. They did so empirically and not so infrequently got favorable results, little realizing that they were dilating an organic stricture.

Along this line it should be borne in mind that pain in the urethra, but with negative vulval, urethral and routine cystoscopic findings, is sometimes found on more thorough examination to be a referred pain caused by ureteral stricture. I appreciate the thoroughness of Stevens' paper all the more because some months ago I reviewed pretty thoroughly the available literature on the urethra in order to benefit an elderly lady who had had a very extensive growth removed from the vulva and urethra four years previously. After a number of urethroscopies and cystoscopies I finally relieved her by dilating a left ureteral stricture which had been the cause of a very distressing pain referred to the urethra.

Along with the use of the acorn-tipped bougie I have found palpation of the urethra with the finger to be of distinct value in the diagnosis of localized hard strictures and also of the fairly common chronic urethritis mentioned by Stevens, in which the whole urethral tube is uniformly and markedly thickened and increased in density.

I would like to call attention to a very much neglected but very effective treatment of chronic trigonitis and cystitis coli which conditions are occasionally complicated by a sensitive inflammatory condition of the urethra near the bladder neck. This condition is most frequently caused by a colon bacillus infection either ascending or descending. In my cases I have found it to be more frequently descending. It does not respond to bladder irrigation, although millions of bladder irrigations have been wasted upon these patients. The treatment was devised by Bierhoff while working in Knorrs' Clinic in Berlin, and is called "Knorrs' cauterization." A urethroscope is introduced into the bladder, thus emptying the bladder. A large cotton swab dipped in one-half of one per cent silver nitrate is introduced into the bladder, the urethroscope withdrawn, and the swab withdrawn after it. This procedure admits of a thorough application of the medicine to the diseased urethra as well as the bladder neck. The treatments should take place about every four days, increasing the strength of the silver nitrate one-half per cent each time up to tolerance, and according to result. The treatment may be somewhat painful, and I agree with Stevens, who remarks, "It is advisable to avoid severe pain following treatments." These patients should be given a hypodermic of some analgesic some minutes before the treatment. Neosilvol or argyrol should also be instilled into the bladder following the treatment on account of their soothing effect.

THE INSULIN TREATMENT OF DIABETIC COMA

By WILLIAM H. LEAKE *

(From the Medical Service of the Los Angeles General Hospital)

SINCE the introduction of insulin recoveries from uncomplicated diabetic coma are so frequent that it is not regarded with sufficient concern by some physicians who fail to realize that even with insulin, coma patients recover only as the result of long hours of hard work by the doctors and nurses.

This paper is based on fifty-three cases of diabetic coma treated with insulin at the Los Angeles General Hospital between January 1, 1923, and January 1, 1926. All but seven of the patients were in deep coma on admission or developed it after entering. The seven exceptions were so stuporous that I have classified them simply as coma patients. Joslin's [1] thirty-three patients treated with insulin with two deaths were largely those in impending coma.

The onset of coma may be sudden, but this is unusual except in severe diabetics receiving large amounts of insulin who are deprived of their supply. Patients who develop coma while taking insulin are extremely resistant to treatment. Nausea, vomiting, epigastric pain, restlessness, drowsiness, weakness, air hunger, and headache are the usual danger signals indicating approaching coma. Abdominal pain, nausea and vomiting are frequently met with in children and at times are mistaken for symptoms of an acute surgical condition, as fever and leukocytosis may be present. Wild delirium is occasionally the first symptom of impending coma. Air hunger, hyperpnea or Kussmaul respiration is practically always present in diabetic coma as a very characteristic symptom. A most important sign in the differential diagnosis of diabetic coma is the soft eyeball, first described by Krause in 1904 and more fully by Riesman [2] in 1916. Krause observed it in twenty-two patients with diabetic coma, but failed to find it in coma from other causes. I have observed it repeatedly in diabetic coma; in several instances the intraocular tension could not be obtained with the tonometer. The cause of this phenomenon is not known.

As the onset of coma is often insidious every diabetic should be warned to communicate immediately with his doctor if any unusual symptoms appear.

Treatment—In October, 1923, the following rules for the treatment of diabetic coma were prepared by Dr. Phoebus Berman, medical director of the Los Angeles General Hospital, and, with certain minor modifications, are still in use:

1. Catheterize the patient and examine the urine immediately upon admission.

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2. Warmth is essential; keep the patient warm with hot blankets and hot water bottles.

3. Empty the lower bowel with a warm soapsuds enema; repeat if necessary; avoid cathartics.

4. Force fluids up to 4000 cc. during each twenty-four hours. If the patient is unable to swallow, administer 1000 cc. normal saline by hyperdermoclysis every six hours (at the present time we often give 5 per cent glucose solution with insulin by this route). Proctoclysis of normal saline or of glucose and soda may be given freely.

5. Administer one unit of insulin per pound of body weight; one-half of the amount is given intravenously, one-half subcutaneously. In patients who are not in deep coma the report of the initial blood chemistry should be obtained before the second half of the insulin is given. If the second blood examination (four hours after the initial dose of insulin) shows that the blood sugar has not changed or has increased, another subcutaneous injection of insulin should be given (one-half the initial dose). The blood sugar and plasma carbon dioxide combining power are determined every four hours. The degree of acidosis, the patient's general condition and the duration of the coma should be taken into consideration in giving additional insulin. Ordinarily the amount of insulin administered up to this time (1½ units per pound of body weight) is sufficient for the first twenty-four hours.

6. Administer approximately one gram of glucose per kilogram of body weight during the first twenty-four hours. If possible, give by mouth in the form of orange juice, which contains 10 per cent carbohydrate. If the patient is unable to swallow, a proctoclysis of 5 per cent glucose should be given (we are now using 5 per cent glucose solution containing insulin by hypodermoclysis). When a large amount of insulin is used it is thought safer to give more glucose than the amount suggested above, but it should be remembered that in ordinary cases of diabetic coma large quantities of insulin are tolerated without marked reduction in the blood sugar.

7. If the patient remains in coma at the end of twenty-four hours following the first administration of insulin, and the blood sugar is still high, the original dose of insulin may be repeated, dividing it into two or three injections during the next twenty-four hours. The blood sugar should be determined every four hours and symptoms of hypoglycemia should be looked for at this time.

8. As children appear to be more susceptible to insulin, give smaller doses. A relatively small amount occasionally produces a marked reduction in the blood sugar.

9. Many coma patients will continue to be sleepy and drowsy for several days after the acidosis has disappeared and after the blood sugar has been reduced. Insulin should be given to these patients with caution as a hypoglycemic state may be brought about easily.

10. Nourishment to the comatose patient is supplied in the form of orange juice. As soon as the individual is able to take food by mouth a balanced diet of approximately 1000 calories per twenty-four hours is furnished. The diet should be of small bulk, consisting mainly of milk, cream, cereal, butter, and orange juice.

Some of the first coma patients in this series were given large quantities of sodium bicarbonate by mouth and by rectum, but now we are content with the administration of not more than 15 to 25 gm. of sodium bicarbonate during the first twenty-four hours. This is the dosage suggested by Starr and Fitz,[3] who have shown that in certain patients with diabetic coma the acidosis may be due to organic acids other than the ketone group which do not respond to insulin but disappear under alkali therapy.

During the past year and a half at the Los Angeles General Hospital insulin has been administered by the subcutaneous route, except in the most severe cases where the initial dose was given intravenously.

Recent literature indicates that the majority of investigators are in favor of fairly large doses of insulin. Joslin,[4] in 1923, advised against more than ten units of insulin as an initial dose in coma cases, but more recently [5] he advocates larger amounts. He states that, "We never intend that a patient at the New England Deaconess Hospital shall come up to within two hours of death from coma without having received at least 150 units of insulin in the preceding hour." I have given in several instances 100 units as the initial dose. In order to prevent any untoward effects from such large amounts it is the custom at the Los Angeles General Hospital to use approximately 1 gram of glucose to buffer each unit of insulin. Campbell and Macleod [6] are of the opinion that carbohydrate thus administered is more efficient as an antiketogenic agent than glucose derived from glycogen in the tissues and from the protein breakdown. The glucose is given for this purpose as well as to prevent possible hypoglycemia.

Of my 53 patients 29 were males, 24 females. The youngest patient was 2½ years, the oldest 67 years. Table I shows the age incidence by decades. Twenty-eight patients were in deep coma on admission and 18 developed coma after entrance to the hospital. There were seven "borderline" cases, so called because these patients were in an advanced state of acidosis and were extremely drowsy. The patients manifesting signs of severe acidosis, moderate air hunger and drowsiness, but who could answer questions are not included in this discussion.

Thirty-seven (69.8 per cent) of the 53 patients died, 16 (30.2 per cent) recovered. Seven patients who entered the hospital in deep coma regained consciousness but died of other complications. The cause of death in the majority appeared to be sudden heart failure. The seven patients termed "borderline" recovered. Eight patients died within twelve hours after admission. Twenty of the 37 patients who died had grave complications, which were discovered at the time of examination or at necropsy (Table II).

The highest blood sugar noted was 625 mg. per 100 cc. of blood, the lowest 181 mg. Two patients had extremely low plasma carbon dioxide combining power—9.9 volumes per cent; one recovered.

One patient was admitted on three separate occasions in coma, recovery ensuing each time; another was admitted twice in coma, recovering each time, and one, a child of 2½ years, died a few hours after his second admission for diabetic coma.

Of the eleven patients who had been under insulin treatment prior to the onset of coma only five recovered, thus confirming the statement made above that severe diabetics who are deprived of their insulin may go into coma very rapidly and that they are singularly resistant to subsequent insulin therapy. The smallest quantity of insulin required to restore a patient to consciousness was 15 units in a child 2½ years of age. Many of the patients who received large doses of insulin remained stuporous for several days, although the acidosis disappeared within a few hours. In the first few patients insulin was administered with undue caution. It is now a well-known fact that so long as the insulin is buffered with sufficient carbohydrate unlimited quantities may be given. I have found it unnecessary to administer

more than 200 units to the average coma patient during the first twenty-four hours, but I would not hesitate to give five times that amount if indicated.

In many diabetics receiving large doses of insulin the blood sugar tends to remain at a high level although the urine is sugar free. Attempts to lower the blood sugar by increasing the dose of insulin may be followed by a moderate reaction, although the blood sugar does not fall below 100 mg. per 100 cc. of blood. Campbell and Macleod [6] suggest that these reactions may be explained on the basis of protein sensitization, but this is not certain. They suggest also that the speed of change of the blood sugar level may be in some way the responsible factor. Major and Davis [7] have noted these reactions in patients with high blood sugar levels. Guthrie [8] in a recent communication states that these reactions may be overcome and a lower level maintained by re-education of the patient or "cell training." Many of my patients who have been receiving insulin for some time show this high blood sugar level with no glycosuria; all efforts to reduce the blood sugar by increasing the insulin have met with failure because of the reactions which developed.

Lack of space will not permit detailed case reports or graphic charts. Tables III, IV, V, VI, and VII show the laboratory data of five patients of this series. Patient No. 192-960 was discharged on a diet of P. 50, C. 70 and F. 205, with *no insulin,* the only patient of those recovering in whom it was found possible to discontinue insulin. Patient No. 201-942, discharged February 2, 1923, receiving 30 units of insulin daily and with a blood sugar of 181 mg. is at the present time receiving 92 units of insulin a day, the blood sugar ranges between 222 mg. and 285 mg. and the urine is sugar free. This patient is unable to tolerate 95 units of insulin without showing a reaction.

SUMMARY AND CONCLUSIONS

1. Fifty-three patients with diabetic coma are reported. The high mortality of this series is explained partially by the large number of grave complications, the long duration of the coma in many, and the failure of several patients to receive treatment for diabetes before coma developed.

2. The symptoms of approaching diabetic coma are often misleading. Diabetics should be warned to communicate immediately with their physician upon the appearance of any unexplained symptom. Abdominal pain, nausea, vomiting, fever and leukocytosis, especially in children, may lead to the diagnosis of an acute abdominal condition.

3. Decreased intraocular tension is a very important sign in the diagnosis of diabetic coma. It is practically always present.

4. In addition to the liberal use of insulin, buffered with carbohydrate, the treatment of diabetic coma consists mainly in forcing fluids, warmth, elimination, and stimulation with caffein sodium benzoate and digitalis. Not more than 25 gm. of sodium bicarbonate should be administered daily.

5. Diabetics develop coma rapidly when deprived of insulin. Coma in these individuals is apparently more resistant to insulin therapy than in patients who have not received insulin previously.

6. Insulin is not infallible. Coma patients recover only as the result of strenuous hours of hard work by the doctors and nurses in attendance.

7. Insulin is an extremely potent drug. In the hands of those familiar with its action it often saves lives, but if used carelessly the results may prove disastrous.

TABLE I

Age incidence by decades: 1st decade 4; 2nd decade 6; 3rd decade 9; 4th decade 11; 5th decade 11; 6th decade 7; 7th decade 5.

TABLE II

Complications in the fatal cases: bronchopneumonia 3; carbuncle 2; pulmonary edema 2; septicemia 1; gangrene (amputation) 1; ruptured pyosalpinx—general peritonitis 1; ethmoidal sinusitis with brain involvement 1; cardiac decompensation 1; multiple abscesses of arms (probable septicemia) 1; intestinal obstruction 1; auricular fibrillation 1; miliary abscesses of left kidney 1; bronchopneumonia—empyema 1; bilateral hydronephrosis and hydroureter 1; gangrene and pneumonia 1; and pulmonary tuberculosis 1.

TABLE III

No. 192-270. Age, 2½ years

Date	Days in Coma	Urine Sugar %	Blood Sugar Mg.	Insulin Units
July, 1923				
3	3	1	625	15
4		0	377	7
5		0	400	7
6		0	333	9
7		0	333	9
8		1		14
9		0.5	333	15
12		0		16
15		0	250	16
19		0	200	16
23		0	166	16

TABLE IV

No. 190-602. Age, 43 years

Date	Acetone	Urine Sugar %	Blood CO_2 Vol. %	Blood Sugar Mg.	Insulin Units
May, 1923					
28	0	2.5		400	0
Patient in hospital on a diet preparatory to perineorrhaphy					
Nov., 1923					
10	1+	trace	61	200	35
11, day after operation	4+	present	57		60
12	1+	0.5	—	—	85
13	0	0	86	117	125
14		0	84	142	35

This patient died of pneumonia. She became comatose shortly after operation, but responded readily to insulin. She received no insulin prior to the perineorrhaphy. Coma developed again a few hours before death, but there was no evidence of acidosis at this time.

TABLE V

No. 192-384. Age, 22 years

Date	Urine Sugar %	Blood Sugar Mg.	Insulin Units
July, 1923			
5	Semi-comatose on admission. Patient went completely into coma twelve hours later.		
6	3.3	363	150
7		347	80
8		333	80
9		285	80
14		400	80
17		142	80

Patient discharged July 26, 1923. Diet P. 45, C. 65, F. 152. Insulin, 80 units daily.

TABLE VI

No. 192-960. Age, 33 years

Date	Time in Coma	Acetone	Urine Sugar %	Blood Sugar	Insulin Units
July, 1923					
18	6 hrs.	2+	2.5	400	80
19		1+	2.5	400	80
20		0	2.0	500	140
23					
30		0	1.0	333	50
August 6		0	0	182	10
23		0	0	118	0

Discharged August 23, 1923. *No insulin.* Diet P. 50, C. 70, F. 205. This is the only patient in the series in whom insulin was discontinued.

TABLE VII

No. 201-942. Age, 22 years

Date	Acetone	Urine Sugar %	Blood CO_2 Vol. %	Blood Sugar	Insulin Units
January, 1924					
11	3+	3.3	10	400	150
12	3+	0	29.6	400	85
14		0.95	39.5	307	60
15		0.8	40.4	285	30
16		1.3	30.9	250	30
17		0.6	42.4	285	30
18		0.5	40.4	285	30
19		0.6	40.4	285	36
21		0.4	48.1	285	36
22		0.2	50.0	222	36
23	1+	0.4	50.0	222	45
24	0	0.08	50.0	200	45
28	0	0.09	—	181	36
Feb. 1	0	0.08	—	181	30

Discharged February 2, 1923. This patient has been under observation in the diabetic clinic during the past three years. He remains sugar free with a blood sugar of 250 mg. He receives 92 units of insulin daily. Any attempt to lower the blood sugar by increasing the insulin produces severe reactions.

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Large scale production, far-flung advertising, and the widespread distribution of goods and ideas have drained something of the color and flavor from American life. Men no longer react individually to conditions as they did in the isolation of the farm or the mine but respond to them under full cognizance of what their fellows are believing and feeling. They think alike, act alike, desire alike. It is a truism that deviation from the usual is nowhere more frowned down upon than in democratic America. Conformity is the price of respectability, and eccentricity is the deadly sin. As nowhere else in the world fashions and fads tyrannize in America.—*Saturday Review of Literature.*

Under the new Medical Practice Act of New York, podiatrists are forbidden to call themselves doctors, even though they follow this designation with the qualifying term of their craft. It is also prohibited to any but licensed physicians to use the expressions "foot specialist," "surgeon," "pedic surgeon," "orthopedic surgeon," or "orthopedic specialist."

PERFORATED ULCERS OF THE DUODENUM

TREATMENT BY HORSLEY OR MAYO PYLOROPLASTY

By EDMUND BUTLER* AND EVERETT CARLSEN

Simple closure is the rational procedure in perforations of acute duodenal ulcers. Such ulcers have existed only a very short time, and the patient often does not give a history of distress previous to perforation. Possibly about 20 per cent of perforated duodenal ulcers come under this classification.

Obstruction seldom follows closure of a perforated duodenal ulcer, therefore it is unnecessary to do a gastroenterostomy because of the fear of this complication.

The surgeon that occasionally encounters a perforated ulcer should be satisfied to do a simple closure.

Perforation of a chronic duodenal ulcer, if seen early, should be treated the same as perforating ulcers. If the induration is great and the patient's condition good a gastroenterostomy is advisable. If there is slight induration and the gastric wall in the region of the pylorus and the antrum is not too much indurated or thickened, then a pyloroplasty is indicated.

DISCUSSION *by R. W. Wilcox, Long Beach; Clinton D. Collins, Fresno; John Homer Woolsey, San Francisco.*

PATIENTS suffering from perforated duodenal ulcers present themselves for treatment in three stages. First the stage of contamination, secondly the stage of peritoneal reaction, and thirdly the stage of progressive peritonitis. During the stage of contamination continuous severe pain in the epigastrium and a board-like rigidity make a diagnosis of perforated ulcer most likely. During the stage of reaction the pain becomes less severe and the rigidity, although present, is less marked, the patient believes himself to be improving, thus the correct diagnosis may not be suspected and the decision to wait a few hours is frequently made. During the stage of progressive peritonitis it is difficult without a most carefully taken history to make a correct diagnosis. The diagnoses of cholecystitis, pancreatitis, diverticulitis, appendicitis, pyelitis and, in the female, pelvic inflammation must be ruled out.

The operative procedures depend on the stage in which the patient first comes for treatment. In the stage of contamination our procedure should be the same as though we were dealing with a perforating ulcer. The presence of a perforation within the first three or four hours should not deter the surgeon from doing a pyloroplasty or a gastroenterostomy. In the stage of reaction the operative procedure must be limited and simple closures and possibly pyloroplasty may be considered, most assuredly not a gastroenterostomy. During the stage of progressive peritonitis simple closure of the perforation is all that reasonably should be done, with pelvic drainage added in the latter stages. Drainage is not used in the stage of contamination or reaction, or in the early stages of peritonitis. Flushing of the peritoneal space should not be attempted.

The advice of some of our leading surgeons is confusing to one not sufficiently acquainted with this type of pathology to have developed considerable individual judgment. Guthrie summarized answers to questionnaires on the subject of perforation of duo-

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